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THE IMPORTANCE OF OCULAR EXAMINATIONS AFTER THE FOURTH DECADE OF LIFE*

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In selecting a subject from so specialized a field of medicine as ophthalmology to present before a general medical body which has in its membership a number of distinguished eye physicians, the essayist must choose a subject which, while addressed to the larger number, may still have some interest for the ophthalmologist. I hope I may not altogether fail in my endeavor to meet these requirements.

In the earlier decades of life the eye, like other organs of the body, is subject especially to acute inflammatory conditions. As such affections are usually accompanied by pain, redness, and at times rapid lowering of the vision the person so affected is conscious of his trouble and at once seeks advice. After the fourth decade, however, frank inflammatory conditions are less frequent, but insidious inflammations and degenerations which are frequently without subjective symptoms are more common and creep on unnoted by the patient. Hence the necessity for ophthalmoscopic examination at this time of life.

Perhaps nowhere else in the body are conditions more favorable for the observation of pathology in the living tissues than in the interior of the eye. Here, with the ophthalmoscope, we see under considerable magnification a transparent sheet of nerve and connective tissue, the retina; overlying but not hiding a structure almost entirely composed of blood vessels, the choroid. The slightest alteration in the transparency of the retina, the result of edema, exudation, coagulation-necrosis of the ganglion cells, or hemorrhages, becomes apparent at once; and in the choroid, sclerosis, atrophy of the vessels, and hemorrhages are plainly visible. The vessels of the retina and choroid are subject to the same changes as vessels elsewhere in the body and we easily recognize with the ophthalmoscope spasm of the vessels, embolism, thrombosis, aneurism, periphlebitis, periarteritis, endarteritis, angiosclerosis, and changes in the quantity and quality of the blood contained in the vessels, especially polycythemia, anemia, hyperemia, pernicious anemia, and lipemia retinalis (Jackson). In this category are mentioned only conditions which, with one or two exceptions, occur with greater frequency after middle life, so as to emphasize the importance of an ophthalmological examination at this period.

One of the annoying tell-tale effects of increasing years is a lessening of the power of altering the focus of the eye which enables one to see at reading distance as well as at far distance. The annoyance caused by this change comes earlier to the far-sighted and in general later to the near-sighted, than to one who has a normal focussed eye for distance.

The ability to alter the focus of the eye for varying distances is called the accommodation of the eye. It is brought about by the power of the reading muscle to change the shape of the lens, contained within the eye, which in early life is soft and plastic. With age the muscle action weakens, the nerve innervation to the muscle lessens, and the lens substance and capsule become more rigid so that the change in the focus cannot be fully effected. These changes are so constant that at about the age of 45, unless the eye be near-sighted, it is no longer possible to accommodate the eye in order to enable one to read ordinary print at a convenient distance without effort and fatigue. This is called presbyopia or old sight. Patients will at times tell with con-

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siderable pride of the remarkably "strong eyes" of a parent who at an advanced age was able to lay aside glasses and read with the naked eye, little realizing that this supposed proof of "strong eyes" is quite the contrary and is but an evidence of beginning cataract. In most cases the first stage in the development of cataract is one of swelling of the lens which causes the eye to become near-sighted.

As the fourth decade is the time of life when more serious changes occur in the eye itself and when a careful examination of the inside of the eye may reveal the existence elsewhere in the body of organic diseases, one should consult a physician specializing in diseases of the eye to obtain the proper reading glass so that he may take the opportunity at the same time of having a thorough examination made of the eyes.

While cataract must in most instances be looked upon as a local disease, yet the fact that it is unusual for it to develop before the fifth decade shows that age is a strong contributing factor and we usually speak of this type as senile cataract as though it were just as much an accompaniment of old age as gray hairs, brittle nails, etc.

Cataract is a loss of transparency of the crystalline lens. This may begin in the periphery of the lens in localized areas and not interfere with the vision for years, or it may begin in the centre of the lens and early in its development interfere with the vision. The former type is the more common. It always affects both eyes but does not as a rule develop at the same rate in both, so that one eye is usually ready for operation while the other still retains some sight. It is no longer considered necessary to await the maturity of the cataract before operating. The cataract should be removed as soon as the patient is incapacitated for his life work. If the patient is advanced in years the extracapsular operation may be done but if the patient is in the earlier decades of life the intracapsular method is preferable.

If cataract is detected in its early stage can it be absorbed or held in check? As yet we have no means of bringing about its absorption, but attention to the hygiene of the eye, to the general physical condition, to diet, and to the wearing of proper glasses may have some retarding effect on its advance.

Cataract sometimes develops as a complication of diabetes. It is always bilateral and develops usually earlier in life than the senile type. The cause is not known, but it is not due to the presence of sugar in the aqueous.

Dinitrophenol may produce cataract and many cases of cataract have resulted from the indiscriminate use of this chemical for the purpose of reducing weight. As it occurs at about the same age period as diabetic cataract, it is well to have this cause in mind when a patient has cataract of rapid development. It may be of interest to you to know that while post-operative complications are more frequent in diabetic cataract, the prognosis is, on the whole, favorable. Especially is this so since the use of insulin.

Of all diseases of the eye the one most dreaded is glaucoma. This is usually described to the laity as a "hardening of the eyeball." What it really is is an increase in the hydrostatic or fluid pressure of the eye. If you press the normal eyeball with your fingers you have a sense of its dimpling. As the inward tension increases it requires more and more pressure of the fingers to accomplish this, and in severe cases of glaucoma it connot be done at all. In other words the eye has become "stony hard." The tonometer is used for a quantitative estimation of the intraocular tension.

In general terms it may be stated that the reason for the increase in the internal fluid pressure of the eye is that there is some obstruction to the outflow of those fluids which are normally formed within the eye to nourish it and also keep it spherical shape.

Glaucoma is preeminently a disease of old age. The incidence increases with each decade, but the disease is rare before the fifth. It occurs in an acute and chronic form, but it is only with the latter that we are here concerned. So insidious is this form of the disease that many cases are not seen by the oculist until after the sight of one eye is already lost, or practically so, and that of the other also involved. Could there be any stronger argument than this for the need of a careful examination of the eyes after the fourth decade?

Only a week before last one of the leading surgeons of Philadelphia called for an appointment, thinking he needed a change in glasses, and to my amazement, when I examined his fundus, I saw he had advanced glaucoma, and in the left eye the lower field was entirely destroyed. Yet he was absolutely unconscious of this defect in the lower field of his left eye and was operating daily.

It seems hardly possible that he should be so unobserving. It is probable that in his final operative work he used only the macula which was not involved, and which still had quite good visual acuity. But that is an illustration of how insidious glaucoma is.

The effect of the increased tension of the eye is to destroy by its pressure the most delicate and susceptible portions of the eye, the optic nerve and the retina.

High blood pressure, increased intraocular tension, and hardening of the arteries are terms all too well known to the public, and doctors are not a little to blame for the feeling of apprehension with which a patient learns that his blood pressure or intraocular tension has gone up; surely at a time when "ignorance is bliss....."

There are those who claim to recognize early increased blood pressure in the retinal circulation but the signs are unconvincing, and the use of the ophthalmodynamometer is the only certain though somewhat difficult way of determining it. The practical value of the instrument is yet to be demonstrated, though it has been said to have led to the diagnosis of increased intracranial pressure before the more certain sign, that of choked disc, was present.

When it comes to recognizing the hardening of the vessels of the retina we are on more certain grounds. One of the early effects of sclerosis of the vessels is the excitation of spasm. When this occurs in the central artery of the retina it gives rise to very definite subjective and objective symptoms in temporary loss of vision and ischemia of the retina. In some cases the spasm leads to permanent obstruction by the formation of a thrombosis. At about the same stage of the sclerosis the systolic impact on the stiffened vessels may cause a rhythmic lateral displacement of part or the whole of the retinal arterial tree on the plane of the retina, the so-called locomotion pulse. This is but infrequently observed.

Some years ago we had the opportunity, through the courtesy of Dr. Harbridge, who was practicing in Chester, to see one of these cases of spasm of the central artery. This man was a produce dealer, and was leaning over a basket arranging the apples—I suppose with the largest ones on top—and he suddenly lost the vision of one eye.

It was a temporary failure, but Dr. Harbridge saw him and brought him at once to Philadelphia, and several of us had an opportunity of seeing him. He had quite a series of spasms. The first thing noticed was that there was a closing in of the field of vision from the periphery towards the center. At that time the pupil was also slightly dilate. If the other eye had been closed, it probably would have dilated at a maximum.

There was then, with the ophthalmoscope, visible a spasm of the arterioles of the retina, not a complete collapse, because that rarely occurs even in obstruction, and then, after just a brief time, vision would slowly return, the spasm would be relaxed, and the vessel would refill. Only on one occasion did it last long enough to produce a slight clouding of the retina.

I followed this patient for many years, and while he developed a considerable degree of sclerosis of the retina vessels, he never had another series of spasms.

As the walls of the retinal vessels are normally transparent, the slightest departure from this quality changes their appearance. As a deposit of hyaline or glassy substance in the walls of the vessels early occurs in arteriosclerosis, the walls of the vessels now give back a bright reflection of the light thrown into the eye. The thickening of the walls causes the vessels to become tortuous and firmer, especially the arteries. When therefore an artery crosses a vein it compresses the walls of the vein and as the artery is now opaque the underlying portion of the vein is no longer visible. The distal portion of the vein is slightly distended. When a vein crosses an artery the vein is usually to a degree flattened, though it may arch forward. The vein is frequently diverted from its course

as it crosses the artery. At such a crossing a localized edema may occur. The arterioles, the branches of the central artery of the retina, show increased tortuousity.

The picture of advanced retinal angiosclerosis with high blood pressure added is edema of the optic nerve head, scattered small glistening hard exudates about the central area, and hemorrhages. It is generally believed that retinal hemorrhages are always indicative of hypertension, and are not seen in pure arteriosclerosis. In a recent article Wagner states that a condition to which he gives the term malignant hypertension shows a fundus similar to that seen in glomerular nephritis, with a higher degree of arteriosclerosis, with angiospasm and absence of massive exudate, and an incomplete macular figure. Papilloedema is present.

There are other evidences of ocular arteriosclerosis besides the ophthalmoscopic ones; recurrent subconjunctival hemorrhages, fugacious conjunctival edema, painful accommodation, and rarely hemorrhage into the anterior chamber.

The importance of the early recognition of retinal angiosclerosis is impressive when it is made known that the condition of the retinal vessels is almost a sure index of the condition of the cerebral vessels. The converse does not seem to hold true.

Indulgences which in early life are without serious or appreciable injury to the body tissues and functions, in later life either as a result of cumulative action of the toxic agents or of lowered resistance of the tissues, or both, may bring in their train functional impairment or serious organic change. Notable among these excesses are relatively high carbohydrate intake, alcohol, tobacco, etc.

Of the exogenous toxic agents which affect the retina and optic nerve tobacco and ethyl alcohol are practically the only ones which show a predilection for later life. Possibly two factors contribute toward this: cumulative action, and vascular changes. However this may be, it is rarely seen before forty years of age.

Day blindness and inability to see to read, which are the two principal symptoms, come on slowly but the annoying central scotoma usually causes the sufferer to seek advice. The symptoms are loss of vision and the day blindness, a scotoma for color (so-called relative scotoma), slight pallor of the temporal half of the optic nerve head, and occasional retinal hemorrhages. If actual atrophy of the optic nerve has not occurred the withdrawal of the poison is soon followed by a return to normal vision. The association of tobacco-alcohol amblyopia and incipient cataract may cause the toxic factor in the loss of vision to be overlooked, the loss of vision being wrongly attributed to the lens changes.

Mention has been made of the occurrence of cataract in diabetes. It may not be entirely germaine to the subject, but as diabetes is much commoner after middle life than before, the ocular complications are significant and may early suggest the diagnosis. We will at least enumerate them; changes in the static refraction of the eye, cataract, paralysis of the extra and intraocular muscles, retinitis, lipemia retinalis, retrobulbar neuritis, irritis, uveitis, and hypotension of the eyeball.

There are of course many other senile changes met with in the eye such as the macular degenerations, but their early detection is not of great importance, as we can do nothing for them.

Nephritic retinitis is likewise seen, but only in chronic glomerulo-nephritis is it essentially a condition of later life. The concept of the relation between nephritis and retinitis has undergone considerable change in comparatively recent times. These changes date to the researches of Volhard, who demonstrated that in the retinitis of hypertension and nephritis there is a generalized constriction of the arterioles throughout the body in a varying degree, and he states that continued constriction of the arterioles leads to organic changes in their walls. The constriction is variable, and subject to sudden marked changes almost daily.

Volhard recognizes renal arteriosclerosis without renal insufficiency, *essential hypertension*. This condition makes its appearance commonly between the ages of fifty and seventy years. At first transient, it becomes permanent. Generalized constriction of the arterioles is absent, but localized constriction may be present. The retinal arterioles become sclerosed and later the picture earlier in the

paper described as retinal angiosclerosis occurs. The appearance of hemorrhages increases the gravity of the prognosis.

In renal arteriosclerosis with renal insufficiency, *malignant hypertension*, there is a general spastic condition of the arterioles throughout the body and all the organs show a marked degree of sclerosis of the small arteries and arterioles. The retinal picture is that already described as the retinitis of malignant hypertension.

The distinguishing diagnostic difference between the fundus changes of malignant hypertension and glomerular nephritis is that in the former there is marked angiospasm and that exudates are not so numerous.

...At the conclusion of his paper Dr. Zentmayer presented slides loaned by Dr. Walter I. Lillie, Professor of Ophthalmology, Temple University, commenting upon them as follows:

(Slide) Here we have a normal fundus. The relation of the size of the arteries to the veins in the normal eye is about in relation of three to four. You see, there is light reflection on the vessels. The vessels are not tortuous, but they do arch rather gracefully out to the temporal side, forming an arch surrounding the central area.

On the nasal side the vessels assume a much straighter course than on the temporal side.

That is a fundus of a dark complexioned person, shown by the presence of so much pigment in the choroidal intravascular spaces.

(Slide) Here we have another normal fundus, but this is in a blond person, and you see that there is little pigment in the pigment cells of the retina, so that the choroidal circulation is laid bare and shows the system of flat ribbon-like bands, whereas the retinal vessels you see are cylindrical.

Here again you see the general relation of the vessels, and that they are not particularly tortuous.

(Slide) Here we have a beautiful drawing of the so-called angiosclerotic retinitis described by Foster Moore, and, you see, here you have a very great decrease in the diameter of the arteries and the veins, especially of the veins, with the light reflection on the arteries very much more pronounced than it was in the nomal eye that I showed you.

Wherever an artery crosses a vein, especially in the left branch there, you will see it appears as though a segment were cut right out of the vein. You cannot see the vein on either side of the artery because of the increase in the hyaline matter in the walls of the artery.

Scattered through the central area, you see those very hard, sharply cut, glistening lesions, and there is a certain amount of edema at the head of the optic nerve, though not conspicuous. Then, please note the increase in the tortuosity of the vessels, and particularly the branches which go up to the area where those bright spots are, the so-called macular branches.

Then, the smaller arterioles on the disk also show a very great increase in the tortuosity. There is only one cotton wool mass there, right at the lower border of the disk.

(Slide) Now, here you have a picture of glomerular nephritis. I might say that some who are authoritative writers today do not accept these views of Volhard, and they would consider this the ophthalmoscopic picture of a chronic nephritis. You see, here you have a swelling at the head of the optic nerve and an increase in the capillarity.

Then, in the vicinity of the vessels, you see these soft, fluffy areas like absorbent cotton, and then, towards the central area, you see the same condition as was shown in the case of an essential hypertension in the previous picture.

According to present nomenclature, this would probably be called a case of malignant hypertension, the only difference between the two conditions being that in one there is a marked general attenuation of the arteries, that is, in malignant hypertension; whereas this feature is absent in glomerular nephritis.

(Slide) Here is a case of pure arteriosclerosis of the retina. The disk is not in any way damaged, the edges are very well defined, the arteries and veins retain their normal relation of three to four, and in the macular area you see developing a figure which probably will ultimately assume a stellate formation.

(Slide) Here is a classic picture of chronic interstitial nephritis, as the older writers termed it. You see, there is a slight edema at the head of the optic nerve. There are numerous places with these fluffy white cotton wool lesions, and a perfect stellate figure at the macular region. There is a moderate degree of angiosclerosis.

(Slide) Here is a case which the older writers would call the exudative type of nephritic neuroretinitis. The vascular changes are insignificant, but exudative features are very pronounced. There are some hemorrhages, you see, on the left of the disk, extending out towards the macular region, and about these there are those cotton wool patches.

(Slide) Here is one where you see very extensive changes. These are not the cotton wool patches. These are fatty changes resulting from previous hemorrhages. You do not get localized outpouring of blood from the retina unless there has been an actual thrombosis of the vein, so there has probably been an obstruction there following a spasm in the inferior temporal vein, which produced the outpouring of blood, which is not recent, as the blood is undergoing absorption.

(Slide) Here are some of the late changes seen in the arteries. This may be senile; very often it is; and it is here that the perivascular changes are very pronounced, especially periarteritis. You see the white bands on either side of the brighter vessels, which are the arteries, and one branch going off to the left. Superiorly, the vessel is converted into a fibrous cord.

(Slide) Here is a similar condition, but in a more advanced stage, where you have very marked periphlebitis, with formation of new vessels on the disk and numerous hemorrhages.

(Slide) This very beautiful picture is one of sclerosis of the choroidal vessels. You remember, in the eye that I showed you as being that of a blond person, you saw those choroidal vessels, but they were red, and here you see they are converted into white ribbons. Those more solid areas are whirls of capillary vessels, all in the process of sclerosis. There is a slight amount of pigmentation.

(Slide) I spoke of the occurrence of macular degeneration in senile eyes, but did not go into it because nothing can be done even if you do dis-

cover it at that age period. But there is one of the lesions of an area of atrophy, confined to the macular region. You see the exposure of the lesion through complete atrophy of the eye and choroid. There is a fringe of pigment, and beyond that there is a very narrow yellow band of partial atrophy of the choroid. That would be very difficult to differentiate from a congenital macular coloboma.

(Slide) Here you have one in which the lesion is surrounded by this broad band of fenestrated pigment, like a filigree of iron, and you see those two choroidal vessels still persisting in the floor of the lesion. The disk has a somewhat yellowish appearance, and the arteries are entirely too bright, so there is a certain arteriosclerosis also.

(Slide) You see here on the right this cobblestone appearance over in the central area. That is a hyaline degeneration, and this may be observed not only in senile eyes, but in the eyes of young people, and it is accompanied by no disturbance of the function of the eye whatsoever. On the left is a picture which resembles it very closely, except that here you have pigment changes surrounding the individual lesions, and here you have a very marked loss of vision at the macula. That occurs only in senile eyes—the one on the left.

(Slide) This first picture is diagramatic, but it shows the functional stage of angiospasms. You see there in sections of the arterioles—we used to call those arteries; they are now considered all branches of the retinal circulation, except the central arteries—in two places there is a localized spasm of the vessel. This is a very early stage, either of arteriosclerosis or hypertension of the essential kind.

(Slide) These are actual photographs of the fundus and I have never seen better ones. Here we have in contrast the retinitis of benign hypertension and that of arteriosclerosis. Now, on the left you see a hypertensive lesion. There is no edema and there are only a few cotton wool areas, but there are a number of hemorrhages. This would be a rather late stage of essential hypertension because hemorrhages are the added feature of benign hypertension, and give a bad prognosis.

Now, on the left side you see the retinitis of arteriosclerosis. The difference there is that on the right side, where you have no cotton wool patches, you have a very marked vascular degeneration, as shown by the constriction of the veins where the arteries cross them, and the conversion in one place there of the artery into a fibrous band, and then the formation of a whirl of new vessels in the lower part of the fundus. The essential difference between those two plates is the absence of hemorrhages in the arteriosclerosis, and that the veins and arteries still hold their proper numerical relation of about four to three; whereas, on the other side, there is a general attenuation, you see, of the arteries. They are all more or less reduced in size.

(Slide) Here we have a later advanced stage of the first picture shown, where we had a localized spasm, and here we have a more or less generalized spasm of the arterioles. It is certainly in a very early stage. The one on the right perhaps shows a little more marked constriction of the arteries or arterioles than the one on the left, because it is in a very early stage.

(Slide) Here we have an early picture of malignant hypertension. You have the edema at the head of the optic nerve, though moderate; you have the attenuation of the arteries; and you have some slight degenerative changes in the central area of the retina. In the left eye there are some cotton wool patches forming. This is a mild degree of malignant hypertension, connoting a general spastic condition of all the arterioles throughout the body, and a beginning sclerosis of the vessels, especially of the retina.

(Slide) Here there are contrasted—this, of course, is diagrammatic—the differences between the retinitis of malignant hypertension and the retinitis of nephritis. You see, the two pictures are very similar. The nephritis has the macular star completed, and the one of malignant hypertension has only a few of the rays or spokes. That is of very little significance. In the retinitis of nephritis the arteries are not as constricted as those in that of malignant hypertension. There is really very little difference. You will notice this snow bank appearance around the head of the optic nerve in both cases.

(Slide) Here is the so-called advanced malignant retinitis or malignant hypertension, a very marked papilloedema, constriction of the arteries, massive exudations, and hemorrhages.

1930 Chestnut Street.

DISCUSSION

CHAIRMAN LAMOTTE: Dr. Zentmayer, I would say that that is a crackerjack paper, and a most excellent and instructive presentation, full of valuable information of use to any practitioner.

I would just like to emphasize the importance of the diagnosis of chronic glaucoma and cataracts, because I have seen or know of too many cases where sight has been lost by people who had been going to optometrists.

As Dr. Zentmayer told you, or inferred, the cataract has no symptoms, no pain, and neither has chronic glaucoma. The eye does not get red, and these people very often go and have glasses changed, with the sight in their eyes decreasing until they are blind, and then they are told to have their cataracts taken out. By that time, the other eye has far advanced disease, and very often is hopeless.

I have often thought that many physicians just do not realize the importance of the differential diagnosis of those two conditions.

Does anybody care to ask Dr. Zentmayer any questions, or discuss anything? You can ask him anything about the eye and he has the information right on the end of his tongue.

DR. R. R. TYBOUT (Wilmington): Years ago I was in the service of a Dr. Charles A. Oliver, and I had access to Dr. Zentmayer's services, who was in one of the clinics there. I felt that I learned a great deal in Dr. Zentmayer's clinic. Therefore, today I come in the attitude of a student. I made every ef-

fort to get here, in the attitude of a student, to learn more, and I not only had my memory refreshed, but I have learned still more.

I think it was a most excellent plan of Dr. Zentmayer's to show the normal fundus. In my talks with the internes of the hospitals in which I am in service I always emphasize the importance, when they are using the ophthalmoscope, of studying the normal fundus as often as there is the opportunity. They do not pretend to be, or maybe do not intend to be, ophthalmologists, but it is most important that they know what the normal fundus looks like, with its occasional anomaly. If they are familiar, then, with that picture, any gross or even subtle pathology is very helpful to them.

In that attitude of learning I would like to ask Dr. Zentmayer what his opinion is in reference to malignant, or various stages of hypertension, as to the formation, or the causation, rather, of chronic glaucoma and the beginnings of cataract.

This is based on Magitot's exposition of the metabolism of a transparent media of the eye by osmosis through the blood vessels, the arterioles. It would seem to me that if the arterioles are as supposed to be, we would then find interference with the osmosis of the vessels, and their balance of osmosis in the nutrition of their clear media, which might cause an interference in the nutrition of a lens as well as in the drainage of the eye.

Another point that has always been somewhat of a problem to me is why, if we have a case of glaucoma, Doctor, and the case is either subacute or acute glaucoma, we have such a very shallow antechamber, and we either do paracentesis for temporary relief or we do an iridectomy, and so on.

I would like to know if it may not be possible that there is an edema of the vitreous humor.

DR. HUPER: I wonder if any data exists in regard to the possibility of vascular changes in cases of cardiac hypotension. I am referring to persons having a prolonged hypotension, over months.

DR. ZENTMAYER: As regards the question of the vitreous humor, in causation of glaucoma, of course there are those who believe that glaucoma is due to volumetric increase in the vitreous humor. This has recently been brought up by Dunn of Virginia, and it is undoubtedly a factor in the causation of the very shallow anterior chamber that you meet with in acute or chronic glaucoma.

In regard to the cause of increased intraocular tension, aside from volumetric increase, that depends on whether you side with those who believe that the humors of the eye are produced by a dialysis of the blood plasma, or whether you believe with Collins that the ciliary body actually secretes the aqueous humor. That contention is supported by experimental work of Adler.

In regard to hypotension, I am not familiar with hypotension, except as an essential disease occasionally seen in children, a temporary condition, sometimes met with in inflammations of the anterior segment of the eye in children. Hypotension is seen in the late stages of diabetes. I am really not in a position to say that you could make a diagnosis with the ophthalmoscope of the condition of vascular hypotension.

SIMPLIFIED DIABETIC MANAGEMENT: A NEW REGIME*

JOSEPH M. BARSKY, M. D. and
CHARLES S. LEVY, M. D.**
Wilmington, Delaware

Diabetes mellitus, the sugar sickness of the ancients, was first recognized as early as the first century. Galen, due to the polyuria, thought the kidneys merely allowed fluid to run through. In India, the sweetness of the urine had been recognized for many centuries before Dobson, in 1775, obtained sugar by evaporation. Chevreul, in 1815, proved this sugar to be the same as that in grapes. Very little further was done until von Mering and Minkowski, in 1889 and 1892, produced a diabetic state in dogs by extirpation of the pancreas. This was the foundation which led to the work, in 1921, of F. G. Banting and C. H. Best, with the chemical assistance of J. B. Collip working in the clinic of J. J. R. MacCleod. They proved the possibility of preparing an extract containing a substitution hormone called insulin. In 1925, J. J. Abel of

*Read before the Medical Society of Delaware, Wilmington, October 12, 1937.
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Johns Hopkins isolated crystals of insulin and suggested its chemical composition. Due to the wide prevalence of the disease and the modern accuracy in diagnosis, diabetes mellitus is assuming an important place in modern medicine.

As we glance over the history through the centuries it becomes apparent that the treatment of diabetes can be divided into three areas: the pre-insulin, before 1921; the Banting era from 1921-35; and the present, the Hagendorn era, 1935-36.

With the advent of protamine insulin, a combination of insulin and fish sperm, the practitioner has at his command a much wider variation in the choice of his treatment of diabetes mellitus. We feel we have entered a new regime. However, close cooperation still must exist between the physician and the patient and the treatment of the disease must follow a carefully preconceived plan. The dietetic management of diabetes is still of primary importance. The choice of diet reflects to a large extent the progress in the treatment of the disease. In the pre-insulin days the high fat, low carbohydrate diet was of necessity in vogue. Since insulin there has been a variation in opinion, some adhering to the low carbohydrate high fat diet, others advocating a medium carbohydrate and still others a high carbohydrate and low fat. With all the disagreement one fact held apparent, a low caloric diet was generally conceived as being advantageous, authorities holding to the truism that a fat diabetic was a bad diabetic. With the discovery of protamine insulin a change in the dietetic conception took place and now the high carbohydrate, low fat, low caloric diet is generally the diet of choice.

The old method of arriving at the dietetic prescription in which the proportion of carbohydrates, proteins and fats were reached by arithmetical formulae has given way to the newer and simpler method where an arbitrary number of grams of carbohydrates are prescribed, the proteins arrived at by means of the desired body weight (the old rule of 1 gm. of protein per kilo of body weight still holding true) and enough fats added to arrive at the decided caloric requirement. This more natural method of reaching a livable diet prescription, plus the greater fluctuation in the time of administration of the new protamine zinc insulin has produced a new regime in the treatment of diabetes. With the addition of protamine insulin to the therapeutic armament the clinician now has three weapons in the treatment of those patients requiring insulin who suffer from this disease. He has first, the regular insulin, purification of that discovered by Banting; second, the crystalline insulin; and third, the protamine zinc insulin. These three preparations differ in the rapidity of their absorption and in the rapidity and duration of their effect. Regular insulin works very quickly and exercises its effect rapidly. Crystalline insulin (a purified form of insulin obviating the addition of any foreign substances and thereby eliminating the possibility of an allergic reaction), was reported by Sayhun of Detroit in 1936. Its advantages are that it lowers the blood sugar gradually and has a continued effect over a longer period, as compared to regular insulin. It reduces the possibility of hypoglycemic reactions or shocks and finally results in a diminution in the number of doses required per day. These results were later confirmed by Hugo A. Freund and Sidney Adler, also Altshuller and Leiser, Rabinowitch and others.

Protamine insulin appeared commercially in February, 1937, in the form of protamine zinc insulin, the zinc being added to stabilize the combination. The difference between the commercial protamine zinc insulin and the protamine insulin of Hagendorn is merely the addition of zinc, a non-toxic metal, which permits the stabilization and therefore the commercial use of protamine zinc insulin. Through the kindness of Sharp and Dohme, we were able to secure this product for clinical experimentation in the early part of 1936 and, early in 1937, published our results in some of our ambulatory cases. The promises that this product gave at that time have been more than born out by developments. In our opinion, it is much superior to any other preparation for use in routine treatment.

The time of administration of protamine zinc insulin, whether given alone or in combination with other insulins, is usually half to three quarters of an hour before the meal. This time varies with the individual case and

some of our earlier unsatisfactory results were due to our failure to properly appreciate this fact. Some of our patients take insulin before breakfast and after lunch, some before breakfast and before lunch, and some before breakfast and after dinner; most receive one dose daily, some before, and some immediately after the morning meal.

Its advantages, and we may say that we use it with a high carbohydrate, low fat diet, are:

1. A progressive improvement in carbohydrate tolerance. The average insulin requirement, while at first larger becomes less.
2. Maintenance of desired weight with a fewer total calories.
3. Lessening of the incidence of acidosis.
4. Average lower blood cholesterol.
5. A better tolerance of infections due to a larger glycogen reserve.
6. Better healing power.
7. A general feeling of well being.
8. Lessening of the dangers of avitaminosis. No special foods are required—a more livable diet which makes a sound economic basis for its usage.
9. A slower and more prolonged action of the insulin.
10. A lessened response to dietetic indiscretions. Lessening of the fluctuation in the blood sugar levels throughout the day and less danger of hypoglycaemic reactions.
11. Greater variation in time at which insulin may be administered.

A large dosage may be given at one time due to its slow absorption. In this connection may we point out that through the courtesy of Sharp and Dohme we have been using a preparation of protamine zinc insulin containing 80 units to a c. c. This preparation is not yet commercially available. Our experience has been that unit for unit U-80, regardless of the size of the dosage, whether 10 units, 65 units, or more, on administration produces the same effect as does U-40.

Due to the limited time allotted this paper we do not desire to introduce a great number of case records but wish to quote briefly the following case as an example of the advantages of protamine over the ordinary insulin.

M. A. W. Age 11 yrs. Admitted in coma with acute diabetes 4/20/36. Standardized and discharged 5/7/36, on 15-0-10 regular insulin. Re-admitted 1/22/37 comatose, due to dietary indiscretions. Discharged 1/30/37 on 20 units protamine once daily. Insulin requirements gradually increased due to failure of cooperation to protamine insulin 27-0-18. Re-admitted 8/22/37 with acute enteritis in a pre-comatose state. Discharged 8/27/37 on 50 protamine, once daily, administered immediately after breakfast with diabetes well controlled and no longer a necessity for an evening dose. This is one of our patients who is taking U-80 insulin. Attention is called to the rapid return to normal in a pre-comatose child, suffering from one of the most dreaded complications of diabetes, an acute enteritis.

In coma a disadvantage of protamine zinc insulin is its slow absorption, consequently, it is not as valuable as is regular insulin. Overdosage produces hypoglycemia insidiously. This effect may be unrecognized and the patient apparently rallying from shock may go into shock repeatedly due to the prolonged action of the insulin requiring more constant treatment. These untoward results are not to be considered as disadvantages of protamine insulin, but rather as a failure to properly choose the type of insulin to be employed in a given case. Proper administration of any form of insulin has no disadvantages. The choice of which insulin to use is all important.

No consideration of diabetes would be complete without the consideration of diabetes from the surgical angle. It is our experience that patients standardized on protamine zinc insulin show less fluctuations in blood sugar levels following surgical operations than those standardized on the old regular insulin.

Fowler, Bensley and Rabinowitch reported twenty-five surgical cases successfully treated with protamine insulin. Their results seem to indicate that protamine zinc insulin can be used in cases with and without infection and in cases with or without the presence of acidosis, and that the average control of the diabetic both pre and post-operatively is much better with protamine insulin.

Obstetrical cases do not hold as much terror with protamine zinc insulin as with regular insulin. The pregnant diabetic should be very

thankful, because, whereas frequently, labor was often fatal for the mother, more often to the child, this mortality has been lowered since the use of insulin and now with the new protamine zinc insulin more mothers are saved and we believe the child mortality will also be lowered. It is a generally accepted hypothesis that the death of the child is due to hypoglycemia caused by an overactivity of the child's Isles of Langerhans to compensate for the lack of insulin in the mother. After the birth of the child the amount of insulin manufactured during or by the child is therefore excessive. By preventing the peaks with protamine insulin this danger is apparently decreased.

We would like to quote the following obstetrical case treated with protamine zinc insulin. Mrs. C. M., age 38 yrs. Female. White. Admitted to the diabetic dispensary of the Wilmington General Hospital taking 10-5-5 protamine. Diagnosis of pregnancy was made. The diet balanced and 20 units protamine insulin given once daily. She was kept with a normal blood sugar and a gradually increased insulin dosage until 6/24/37 when 30 units were given (25-0-5). This dosage gradually increased until 8/15/37 when she went into spontaneous labor and was delivered of a normal female child. At the time of delivery she was taking 25-0-10 protamine insulin with a normal blood sugar. Recovery was uneventful, and she was discharged on 8/25/37, insulin requirement being reduced to 10-0-0. Blood sugar on the baby six weeks after delivery was 104. Patient is now taking 18 units of protamine once daily and is very well controlled.

The complications of diabetes are still a great factor. These are divided into three main classes: cardio-vascular, infections, and acidosis. Acidosis in protamine zinc insulin cases properly treated has become a rare complication due to the ability of protamine zinc insulin to handle dietary indiscretions that in former years produced disastrous results, the greater glycogen reserve apparently improving the carbohydrate tolerance of the patient. In our experience diabetics with infections have had much less disturbances of their blood sugar levels once they were properly standardized with protamine zinc insulin.

It is still too early to say what will happen with our cardio-vascular complications but there is less danger of coronary reaction with protamine zinc insulin due to the lessened incidence of shock.

There has been noticed in many clinics an increase in the incidence of peripheral vascular disease. This is probably due to the increased life span of the diabetic making it possible for him to live long enough to develop complications. This circulatory impairment with its menace of gangrene is of grave significance, formerly in the presence of gangrene, particularly with greatly diminished oscillometric readings and with histamine tests showing markedly decreased circulation, a high amputation was always advocated.

J. B. Wolffe of Philadelphia and his coworkers, reasoning backward, recalled that in the days of impure insulin there was less incidence of coronary attacks during shock than with the purified insulin. They theorized that in the extraction of the insulin some product was left behind which would have a favorable effect upon these arteriosclerotic conditions. They produced a preparation, an insulin free pancreatic tissue extract, which they attempted to use in the treatment of cardio-vascular diseases. The next thought was, since it apparently aided in the treatment of diseases of the coronary, why not in terminal artery disease?

For the past five years, we have been working in cooperation with Wolffe and his associates, and we have been employing pancreatic extract (prepared by him and in the past few years prepared in a similar fashion and standardized according to his methods by Grant, under the name of "pancreatic hormone") as a prophylactic measure as well as after the onset of gangrene.

We have found on the administration of this extract a choline-like substance which we call quasicholine is increased in the urine, the significance of this we are still studying.

We make it a rule to question patients closely as to the possibility of circulatory diseases in the lower extremities, and we examine these extremities very carefully for impairment of circulation. Upon the discovery of any evidence of impairment we immediately start the administration of one to two c. c. of pancre-

atic hormone intramuscularly daily, or three times weekly, depending upon the severity of the case. We find quite frequently that following three or four weeks treatment there is usually a marked improvement in circulation and walking capacity.

Some of these cases will be reported shortly in one of the current medical journals.

The future holds the answer to these various problems. We have gone a long way, but until we can assure the diabetic that the incidence of cardio-vascular disease is almost eliminated we have still much work to do. With the prolongation of life resulting from insulin we are now confronted with a new disease. What is the complete future of the diabetic child?

To summarize: our conception of the modern treatment of diabetes is a diet sufficient to maintain the patient at the desired weight, one that is relatively high in carbohydrates and low in fats, with or without insulin, depending upon the patient's individual requirement. We feel that with this type of dietetic formula, and with the addition of protamine zinc insulin to our armamentarium, diabetic management has been greatly simplified. There is no substitute, of course, for the normal pancreas, but insulin is the best we have to offer as yet. We have come a long way since Banting's epoch-making work. Probably in no other disease has medicine made such gigantic strides, but until we are able to assure our diabetics that we can, not only treat his complications but absolutely prevent them, we have still much work to do.

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DISCUSSION

DR. CHARLES S. LEVY (Wilmington): I have assisted Dr. Barsky in the preparation of this paper, and, as he has stated, we have omitted the case histories upon which the results have been based. I want to emphasize this fact: that we are very highly pleased with the results we have obtained with the use of protamine zinc insulin.

The new preparation that Dr. Barsky mentioned, which is the insulin-free pancreatic hormone, has shown some excellent results in our hands. It is especially useful in the few cases that I recall in assisting these arteriosclerotic patients to have a greater walking capacity, which was absent before they started to take this new preparation.

DR. SIDNEY CHAVIN (Wilmington): I would like to ask Dr. Barsky to tell us of some of the early symptoms in the case of a child having diabetes. I mean in the early stages, the early symptoms, not as far as coma.

Another thing I would like to know is whether the pancreas is the only organ that is producing insulin in the normal individual; and also concerning rehabilitation of the various organs producing insulin after some treatment.

PRESIDENT WHITE: Are there any other comments?

I remember when I was a younger doctor than I am today that with diabetic patients, as I recall, we used to feed them all the water they could drink, and they were everlastingly thirsty; we would feed them all the food they could eat, and they were everlastingly hungry. It seems to me that diabetics has gone a long way in these last few years.

Dr. Barsky, would you care to answer those questions now?

DR. BARSKY (Wilmington): The early symptoms of diabetes in a child are no differ-

ent from those in the adult, other than usually they are more acute. Symptoms, of course, of practically all diabetics in the early stages, are digestive: first, hunger and thirst; then polyuria, with loss of weight and loss of strength.

In the case quoted, the child whom we called an acute diabetic, that child had been operated upon for acute appendicitis two months before the onset of the diabetes. At no time during the child's hospitalization—and I personally looked up the record—was there anything to suggest a diabetic state. Yet she was brought into the hospital in coma, with the blood sugar, to the best of my recollection, well above five hundred milligrams, and a CO_2, about twelve.

As to the question of the pancreas being the only organ that produces insulin, we know that insulin is produced by other organs in the body. Insulin can be extracted from various glands, as the thymus and submaxillary salivary glands, the liver, the blood, the muscles, etc., even the urine of normal individuals. We are all aware of these diabetics who are apparently insulin resistant. Every once in so often you find a diabetic who apparently will not respond to insulin. You give them insulin or you do not give them insulin, and the blood sugar level remains apparently the same.

From our concept of diabetes as a pancreatic disease, we do not believe these to be true diabetics, no more than in the case of these fat diabetics who come to us complaining of itching, frequency, thirst, hunger, showing glycocemia and elevated blood sugar levels, who, upon diet alone, with a reduction of weight, eventually are able to practically forget their diet entirely, and show no evidence of diabetes.

Does that answer your question, Dr. Chavin?

DR. CHAVIN: There is one more—about rehabilitation of the various insulin-producing organs after treatment.

DR. BARSKY: As far back as 1926 the teaching was that the pancreas had a definite tendency to regenerate. Whether the fact that the pancreas itself is ordinarily able to metabolize more sugar than we usually take is responsible for this, or whether it is the fact that insulin tides the pancreas over the period of time when it is overburdened, and causes an apparent regeneration in the ability to metabolize sugar, we do not know. We do know, however, that diabetics die, and to the best of our knowledge show us a normal pancreas.

If some other organ, as the liver, is not the cause of the diabetic findings, then apparently we do not know enough pathologically to find the reason in the pancreas itself.

Third Annual Postgraduate Institute Philadelphia, March 28th to April 1st

The large number of advance registrations presages a large attendance of physicians at the Third Annual Postgraduate Institute to be held in Philadelphia, March 28th to April 1st, 1938.

The subject of this year's meeting is "Diseases of the Digestive Tract" and the speakers are all men of national reputation.

Among the features of the session is the opening day luncheon on Monday, March 28th at which the following will be guest speakers—Hon. S. Davis Wilson, Mayor of the City of Philadelphia; Dr. William C. Hunsicher, Director of Health, City of Philadelphia and Dr. David W. Thomas, President Elect of the Medical Society of the State of Pennsylvania.

On Wednesday evening, March 30th, there will be a dinner at which Dr. Frederick J. Bishop, President of the Medical Society of the State of Pennsylvania will be a guest speaker.

Dr. Arthur C. Christie of Washington, D. C., will deliver the J. Chalmers Da Costa oration on "Comprehensive Planning for Medical Care—The Physicians' Responsibility."

EDITORIAL

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THE SURVEY OF MEDICAL CARE

For many years back—long before the Committee on the Costs of Medical Care was invented—the American profession has been harassed and the public bombarded with the most wild and fantastic stories about the inadequacy and the unavailability of the services rendered by the medical profession. When such stories reached the ears of the thinking people, the reaction was generally small or nil—the thing was discounted as representing, generally, a loud-mouthed coterie of persons, propagandists by profession, who had their own axes to grind, and being laymen of the economic, engineering, social service, etc., professions, their strictures anent the medical profession and its work were not taken very seriously, for they had no knowledge of, experience in, or sympathy with the rank-and-file doctor and his work. For the most part, these bleatings represented speciously, a sob-sister appeal to the psychology of the masses, the forerunner of what the present national administration delights in calling "concern for the underprivileged." The significant thing during all those years is that while these protagonists of state medicine, compulsory health insurance and various and sundry other un-American ideas were shouting from the hilltops for the poor, the unassuming rank-and-file doctors were down in the valleys working for the poor, donating services worth millions of dollars a year.

Serene and complacent in the contemplation of a difficult task well done, the medical profession was caught in the maelstrom of a world-wide depression and began to ask a few questions of itself: Why do we work hard day and night and barely earn a living? Why has the general purchasing power sunk so low that medicine is becoming a luxury? What is wrong with business that it cannot pay its workers a living wage? Plunged into a serious plight, these and many more were the questions for which they sought an answer, and at last an authoritative answer is on the way, at least of the medical part of the problem. The major problem, perhaps, is the economic one, concerning which likewise facts are finally coming to light. (Read the editorial which follows.).

Pursuant to a Resolution of the Board of Trustees of the American Medical Association, each county medical society—and there are over 2,000 of them in the United States—and each state medical society is to make a thorough survey (1) to determine the prevailing need for medical and preventive medical services; and (2) to develop the preferable procedure for supplying those needs. This study of the demand and the supply side of medical services will be a laborious one, but it is something that simply must be done, first, because there *are* some people without adequate service; and second, because only with actual facts can the radicals and the propagandists

be silenced and defeated. We want no more glib prattle about "one-half of the people who need medical care cannot get it;" nor do we want any more extemporaneous guesses that "less than 5% of the people who need medical care cannot get it." What we do want is facts, all the facts, and with these in our possession, the medical profession will be able to solve one of the most pressing of our public problems.

The gigantic scope of this study can be glimpsed from the articles in the *Journal of the A. M.A.*, January 15, 1938, Editorial, page 212; February 12, 1938, p. 77B; and March 5, 1938, page 127B. The work will involve every agency that touches sickness—physicians, dentists, nurses, hospitals, clinics, health departments, relief agencies, educational institutions, etc. The mere enumeration of the agencies that must be interrogated shows what a job is before us, but it will be done, and done deliberately and accurately. The Medical Society of Delaware and the New Castle County Medical Society each has a Committee on Medical Economics; it is now necessary for Kent and Sussex Counties to set up immediately the committee that shall have general supervision of the survey in those counties. With these and the national blank forms in hand, the survey in Delaware will soon be under way.

TRUE CAUSE OF LACK OF MEDICAL CARE

Surveys, investigations, studies, statistical reviews, reports and other inquisitions have in recent years been applied to the medical profession with such frequency and regularity that the conscientious medical man is apt to consider such studies as propaganda, issued by someone with an axe to grind and largely of no real benefit to anyone. A recent report, however, of the United States Public Health Service is interesting because for once the medical profession is not accused of inefficiency, inadequacy and selfishness. For once the lack of medical care for a large section of the population is not laid at the door of the medical practitioner.

Since no individual or group is accused by this report and since it does not show the medical profession to be withholding for selfish reasons proper medical service from the public, this statement of the United States Public Health Service will probably not receive wide publicity. Nevertheless, it does contain certain food for considerable thought. A Works Progress Administration grant of $4,000,000 provided funds for the study. A total of 800,000 families, representing 2,800,000 persons, were quizzed by the WPA investigators. Of these families, eighty per cent had incomes of less than $2,000 a year, and forty per cent less than $1,000. The report showed that thirty per cent of illness in these low income families received no medical care. It further showed that the infant death rate among the families with the lowest incomes was 168 per 1,000, as compared to 30 per 1,000 in a group of families of $3,000 annual income.

It is, therefore, quite apparent, that the inadequate distribution of medical service, so long and so loudly decried by the reformers and promulgators of studies on medical care, belongs in the same category as the inadequate distribution of good food, decent lodging, proper clothing and other essentials of human existence. For once it is shown that those able to purchase the ordinary necessities of life are also able, under the present system, to obtain sufficient first class medical service. It is, therefore, apparent, that correction of the evils of inadequate medical service to large groups of the population will be corrected when the general economic situation is corrected. The solution of the problem of medical care for the indigent is no more simple than that of the problems of proper food, proper clothing, proper housing.

Indeed, the report itself states: "It is apparent that inadequate diet, poor housing, the hazards of occupation and the instability of the labor market create immediate health problems." At last the blame falls where it should.

Editorial, *Northwest Med.*, Feb. 1938.

MISCELLANEOUS

Physicians Tour to the A. M. A. Convention

The thought that the forthcoming A. M. A. Convention in San Francisco, June 13th to the 17th, is such a splendid opportunity for a tour of the United States both going out and coming back, has inspired definite action. The cooperation of more than 25 state medical societies has made it possible to arrange a special train tour which will include such outstanding highlights of the North American continent as the Indian Detour, the Grand Canyon, Los Angeles, Riverside and Santa Catalina Island—on the way out to San Francisco. A choice of two return routes is possible, one of which visits the charming cities of Portland, Seattle, Victoria and Vancouver and the beautiful scenic spots of the Canadian Rockies; the second route travels via Yellowstone National Park, Salt Lake City, Royal Gorge, Colorado Springs, and Denver.

There is an all-inclusive price for this tour which includes transportation from home-town to home-town, though the tour starts officially at Chicago on Monday, June 6th, from which point an American Express escort joins the group, as this travel company has been appointed transportation agent and the business details of the trip are in their capable hands.

Let us take a preview of the tour. The first day out of Chicago, racing across the broad, wheat growing face of Kansas, we become acquainted with our traveling companions, physicians from other states, their families and friends, and find ourselves among congenial, like-minded traveling companions. We first leave our train at Lamy, New Mexico, to enter the Indian Pueblo district by motor-coach. We spend a whole day exploring the traces left by a vanished civilization on this continent, visiting Santa Fe, Tesuque, Puye and Santa Clara Pueblo.

The next morning's arrival at the Grand Canyon will remain in our memories forever. The vast chasm, 4 to 18 miles wide from rim to rim gives us stupendous vistas of awe-inspiring beauty, unparalleled the world over. We drive over the famous Hermit Rim Road, skirting the edge of the chasm in the morning, and in the afternoon over the Desert View Road through the Tusayan National Forest and along the Canyon's rim, stopping at Yavapai Point Observation Station for a short, interesting lecture by the Park Naturalist. This drive ends at the Watch Tower, a recreation of the ancient towers erected by the prehistoric inhabitants of the southwest.

The golden, amazing city of Los Angeles is next on our itinerary, and our sightseeing trips acquaint us with its Spanish Quarter and Chinatown, as well as its beautiful environs, including flowering Pasadena. Riverside and its orange empire, its lemon and grapefruit orchards and its famous Mission Inn, is another destination; and then, on our third day in California we sail to beautiful Santa Catalina Island, playground of this land of the sun. And in this delightful manner, a week after leaving Chicago we arrive at San Francisco in time for the Convention. We shall not discuss the interesting time that awaits us at our conclaves, as the object of this article is to describe the pre and post-convention tour. So we turn again to our itinerary after the Convention.

Supposing we had chosen Return Route No. 1. We shall visit Portland, Oregon, famed as the City of Roses, and enjoy as well a drive along the noted Columbia River Highway. Seattle is next, and here we also cover all the points of interest, including both the Lake and Sound districts. Now the Canadian part of our journey begins, and we sail by comfortable steamer to the cities of Victoria and Vancouver, where we do sightseeing. Now a train takes us into the enchanting scenic regions of the Canadian Rockies, and we stop at Chateau Lake Louise, at the lake of the same name—a gem of exquisite color, surrounded by green forests and snowy peaks. Our drives through the heart of the Rockies takes us to Moraine Lake, the Valley of Ten Peaks, Johnson Canyon and finally to Banff, where we make another stopover. After additional sightseeing around Banff, we entrain for Chicago.

Return Route No. 2 takes us to Chicago in a more southerly route. A 3½-day tour of Yellowstone National Park is one of the highlights of this tour. Ranger naturalists conduct our party to the geysers and hot pools, and we feast our eyes on Old Faithful in its hourly eruption. We also see the Grand Can-

yon of the Yellowstone and Mammoth Hot Springs. Salt Lake City is on our itinerary, which gives us an opportunity to visit Saltair Beach on Great Salt Lake, also the Great Copper Mills and Smelters. Our next call is at Colorado Springs, the noted health and pleasure resort. Our travels in the Rockies take us up to the summit of Pike's Peak, to the Garden of the Gods, to Seven Falls, and finally to Denver. This lovely city is a center for outings in the Rockies, and we are soon off on a 65-mile tour of Denver Mountain Parks, including Memorial Museum and Tomb of Buffalo Bill of western fame. From Denver we travel to Chicago.

The above is barely a glimpse of the outline of the tours, but it is hoped that some idea has been given of the enjoyable travel awaiting those physicians and their families and friends, who wish to combine attendance at the Convention with an interesting journey and a happy vacation.

Components of Vitamin B Complex

E. M. Nelson, Washington, D. C. (*Journal A. M. A.*, Feb. 26, 1938), defines the "vitamin B complex" and explains the nomenclature that has been used to designate individual members of the complex. Only two members of the vitamin B complex, vitamin B_1 and riboflavin, have been shown to be chemical entities. Only two members of the vitamin B complex, vitamin B_1 and the P-P factor, have been unequivocally linked with deficiency diseases in man. The demonstrated function of riboflavin in oxidation reduction systems, its wide distribution in living cells, its demonstrated relation to growth of rats and chicks and the development of cataract in rats indicate that it is probably an important nutritional essential for man. Clinical investigations have led to the conclusion that riboflavin is ineffective in the treatment of human pellagra and animal experimentation leads to the conclusion that lack of this substance is not responsible for the development of blacktongue in dogs or nutritional dermatosis in rats or chicks. There is at present no conclusive evidence that blacktongue in dogs as produced on Goldberger and Wheeler's diet is not the analog of pellagra in man. Evidence in accumulating which indicates that a nutritional dermatosis in chicks may be closely related to human pellagra. Elvehjem and Koehn have found that concentrates which will prevent chick dermatosis are also quite effective in curing blacktongue in dogs. Fouts, Lepkovsky, Helmer and Jukes found that the "filtrate factor" of Lepkovsky and Jukes, which had been demonstrated to be preventive of a chick dermatosis, was curative of human pellagra and of blacktongue in dogs. If the observations of Smith which indicate that the P-P factor of Goldberger consists of two or more factors are confirmed, the exact relationships of the deficiency diseases in the dog and chick to human pellagra cannot be visualized. The relation of vitamins B_3 and B_5 to other members of the B complex is not clear. If the nutritional requirements of the pigeon are similar to those of the chicken a reassessment of the importance of these factors will be necessary to catalog them properly with other factors. Since the original work on vitamin B_4 seems to have been disproved and attempts to confirm more recent work on this factor have not been reported and factor W has not been associated with any deficiency syndrome, the importance of these factors is also rather obscure.

Differential Diagnosis of Pain Low In the Back

ARTHUR STEINDLER, in collaboration with J. V. LUCK, Iowa City (*Journal A. M. A.*, Jan. 8, 1938), believes that the allocation of the source of pain due to disorders low in the back offers a distinct advantage for the management of the disorder. This allocation is thoroughly feasible in the large number of cases in which the trouble involves structures supplied by the posterior division of the spinal nerves, because the seat of pain is either accessible to the palpating finger or reveals itself by transmission through the leg test or both. An attempt has been made to show by the procaine hydrochloride test that both local pain and radiation are in causal connection and that radiation may be elicited by an area of local pain as a reflex symptom without being caused by root compression. This theory does not by any means reject true root compression neuralgias as they occur with arthritis or with special pathologic conditions of the lumbosacral level. Proof is furnished that in the large group of cases of the posterior division syndrome such radiation is a reflex phenomenon, because it can be suppressed together with the local pain by the injection of procaine hydrochloride.

Vomiting of Pregnancy

Recently in a case of intractable vomiting of pregnancy in which jaundice had begun to develop, it seemed possible to John M. McGowan, J. O. Baker, Arthur M. Torrie and John Lees, Edmonton, Alta. (*Journal A. A. A.*, Feb. 12, 1938), that muscle spasm of the common bile duct due to obstruction of its lower end might be a causative factor. Glyceryl trinitrate was given after each meal with the result that vomiting stopped and a proposed therapeutic abortion was rendered unnecessary. This experience suggested the following study: A duodenal tube of the Sawyer type was passed into the duodenum according to the usual method. With the patient flat on her back on the x-ray table the duodenal tube was injected slowly by means of a syringe with a suspension of barium sulphate. When a sensation of resistance was transmitted to the thumb, injection was discontinued and a roentgenogram immediately taken. If no resistance was felt, 40 cc. of suspension was injected. Fluoroscopic studies were also made. Results of roentgenograms showed the first and second portion of the duodenum in normal persons to be a curved, sausage shaped structure lying to the right of the first, second and third, or the second, third and fourth lumbar vertebrae and completely filled with barium. Ten minutes following subcutaneous injection of morphine, the second portion of the duodenum seemed to be in a state of spasm. By injecting the common bile duct by means of a T tube, at the same time as the duodenum was injected with barium, it was demonstrated that this duodenal spasm produced a closure of the lower end of the common bile duct and an increase in pressure within the biliary system. Similar duodenal studies were performed on two patients who suffered from severe vomiting of pregnancy. In each case a marked spasm of the second portion of the duodenum was present. The pylorus was relaxed, and reflux of barium into the stomach took place. The stomach seemed to lack tone, as evidenced by the low level of the duodenal tube as it crossed the vertebral column. Following inhalation of amyl nitrite, the second portion of the duodenum was relaxed and therefore restored to normal. Further, the pyloric tone was increased and there was less reflux of barium into the stomach. The gastric tone was improved, as evidenced by the fact that the duodenal tube crossed the vertebral column the length of one vertebral body higher. The duodenal spasm which is found in the vomiting in pregnancy is similar to that which had been produced by the administration of morphine in normal individuals. Morphine had also produced extreme nausea and sometimes vomiting in a few normal individuals. Twelve consecutive patients who suffered from severe vomiting of pregnancy were treated by means of glyceryl trinitrate (nitroglycerin) 1/100 grain (0.0006 Gm.) under the tongue before and after meals. The results were uniformly good. All patients ceased vomiting within two days of the onset of treatment; one patient did not vomit once after the drug was used. It was found that taking the drug ten minutes before meals gave more complete relief of nausea than taking it after meals. No untoward effects were noticed except a transient headache of a few minutes' duration. The patients were instructed to remain in the prone position for ten minutes after placing the tablet under the tongue.

OBITUARY

JAMES L. FRANCE, M. D.

Dr. James Lindsay France, of Wilmington, chief consulting physician of the Delaware Hospital, who retired from active practice about three years ago, died at his home on March 14, 1938, of pneumonia, aged 68 years.

Dr. France was born in Norwich, Conn., July 12, 1870, a son of the late Robert A. and Lydia Lindsay France. He received his education in Norwich Academy and Columbia University, getting his degree of doctor of medicine in 1892. He served as interne at the Sloan Maternity Hospital and in the outpatient department of the Roosevelt Hospital in New York. He served in the Spanish-American War as major-surgeon.

Associated with the Delaware Hospital since 1893, Dr. France served as assistant in medicine and surgery, chief of medical service, president of the staff, and was appointed chief

consultant when he retired from active practice.

Dr. France was a member of the Sons of the American Revolution, the American Medical Association, the Medical Society of Delaware, the New Castle County Medical Society, Philadelphia Medical Club, Union League of New Haven, Conn., Hay Harbor Yacht Club at Fisher's Island, Temple Lodge No. 11, A. F. and A. M., St. John's Chapter, R. A. M., St. John's Commandery, Knights Templar, Gunning Bedford Council, Delaware Consistory, Lu Lu Temple of the Mystic Shrine, Philadelphia, the National Sojourners, and the University Club. In politics he was a Republican.

Dr. France was critically ill about two years ago from a heart condition from which he never fully recovered. Dr. and Mrs. France spent much of their time at their home at Fisher's Island.

Dr. France is survived by his wife, the former Miss Virginia Beeson, whom he married September 3, 1919; one daughter, Mrs. Henry Walter, Tegucigalpa, Honduras, and two grandchildren, Jean France and James Henry Walter.

The funeral services were held on March 17, 1938, at the Chapel of the Wilmington and Brandywine cemetery, with burial in the same cemetery.

BOOK REVIEWS

Synopsis of Genito-Urinary Diseases. By Austin I. Dodson, M. D., Professor of Genito-Urinary Surgery, Medical College of Virginia. Second edition. Pp. 294, with 112 illustrations. Cloth. Price, $3.00. St. Louis: C. V. Mosby Company, 1937.

Dodson's Synopsis admirably fulfills the purpose for which it is intended, a reference work for the busy practitioner and student whose hours are crowded. The second edition includes all the desirable new material that has appeared since the publication of the first edition.

Handbook on Nasal Accessory Sinuses. By Frank L. Alloway, M. D. Pp. 121. Cloth. Kingsport, Tennessee: Kingsport Press, 1937.

The reviewer of this book had his doubts as to the value of only 121 pages on such a large subject as the sinuses and their diseases. There is, however, a lot of meat in this little book. As the author says, part of this work is from his lectures at the Postgraduate Course given at the U. S. Diagnostic Center, Washington, D. C. The matter is concise and reliable, even including operations.

There is a good chapter on the sinuses versus eye disorders, where focal infections and the size of the optic canal in their relations to optic nerve disease are emphasized.

Emotional Adjustment in Marriage. By LeMon Clark, M. S., M. D., Assistant in Obstetrics and Gynecology, University of Illinois. St. Louis: C. V. Mosby Company, 1937. Pp. 261. Cloth. Price, $3.00.

This rather small book is not written as a scientific treatise; it is more or less of a popular nature. It contains information which every physician should know and which is unfortunately neglected in medical training. He discusses sex in a rational manner and attempts to emphasize the fact that the instinct must be accepted in a normal manner and as a part of human physiology. Beyond this he attempts to elevate the instinctive drive to coincide with the cultural level of civilization. He feels that the general practitioner should be able to give advice to young people before they are married. He also emphasizes the correction of physical defects which might interfere with proper marital adjustment. One chapter is devoted to the advantages and disadvantages of various types of birth control. Although many books have been written on this subject, one feels that the author has attacked the problem in a sane manner, although at times he seems to be almost too idealistic. It is a book which the intelligent lay person could understand and one which a physician who has neglected the subject of marital and sexual adjustment in regard to his patients, should read. It is undoubtedly true that many neurotic patients would experience a relief of symptoms if they were taught to accept the sexual instinct in the same manner as any other, and if their fears and feelings of guilt were removed by confidential interview with the physician.

1789—MEDICAL SOCIETY OF DELAWARE—1938

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